Clinical, Electrocardiographic, Echocardiographic Characteristics and Long Term Follow up of Elite Soccer Players with J Point Elevation

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Abstract

Background: J point elevation is common in athletes, nevertheless the influence of structural changes associated with the athlete's heart and its prognostic impact are still debated. Our aim was to investigate the prevalence of J point elevation, the associated clinical, electrocardiographic and echocardiographic characteristics and its impact on long-term outcome in elite soccer players.

Methods and Results: clinical, electrocardiographic and cardiopulmonary exercise test data from 332 male professional soccer players were retrospectively analyzed. For 235 (70.7%) athletes echocardiographic data were also available. J point elevation was defined as an elevation ≥ 1 mm in at least two contiguous leads. Long-term follow-up data were obtained for all athletes, while univariate and multivariable analyses were performed to assess the associated characteristics.

Overall, 118 (35.6%) athletes showed a J point elevation ≥1 mm. At multivariable analysis a significant direct association of interventricular septum thickness (OR 1,224 95%CI 1,014-1,478; p=0,036) and Sokolow Lyon index (OR 1,031 95% CI 1,002-1,060, p=0,033) and an inverse association of baseline heart rate (OR 0.985, CI 95% 0.945-0.993, p=0.011) with J point elevation were observed.

Over a long-term follow-up (median 13,3 years; interquartile range 10,1-17,03 years) no differences in survival were observed between groups (0% vs. 0,01%, p=0.294).

Conclusions: The correlation between J point elevation and interventricular septum thickness suggests a possible mechanistic role of exercise induced left ventricular hypertrophy as the basis for J point elevation. After a long-term follow-up, no significant differences in survival were observed between athletes with and without J point elevation.

Introduction

For more than 70 years since its original description[1], the electrocardiographic pattern defined as “early repolarization” (ER) or “juvenile ST variant”, characterized by an elevated take off of the S-T segment at the J junction of the QRS complex and an upward concavity of the S-T segment has been considered as a normal common variant.

In the general population, its prevalence ranges between 5% and 13% while in athletes rises from 20 to 90%[2-4].

From the second half of the ’90s, a growing number of case reports, case series, observational and prospective studies reported that the presence of various electrocardiographic patterns attributed to ER but defined only by the presence of a J point elevation with or without ST segment displacement, may constitute a potential marker of increased risk of sudden death in otherwise normal subjects, casting a dark shadow on this ECG peculiarity[5-8]. Doubts about its real prognostic impact were emphasized even more during the pre-participation screening of competitive athletes, prompting the need for a broad cardiological evaluation, often including also an arrhythmological consultation with subsequent increase in costs.

The classical ER morphology characterized by the combination of J point elevation and elevated ST segment with an upward concavity has not been

recognized as a marker of risk in the general population[2,3,7,8]. For this reason, the classical ER pattern found in athletes, by inference from the general population[2] and from short-term follow up studies[9], is considered a benign phenomenon. In contrast, evidence of an increased risk for sudden death exists for ER patterns characterized by the presence of a J point elevation associated with a horizontal/descending ST segment. Two main questions remain unanswered: 1) is J point elevation observed in competitive athletes somehow linked to structural changes associated with the athlete's heart or is it a mere electrophysiological phenomenon? 2) does J point elevation really imply an ominous prognosis in competitive athletes?

Recent studies have investigated the prevalence of J point elevation and the clinical, electrocardiographic and structural left ventricular characteristics associated with this ECG finding in various types of competitive athletes (from intercollegiate to Olympic), reporting only short-term follow-up data[2,9].

The aim of this work is to investigate the prevalence of J point elevation, its clinical, electrocardiographic and echocardiographic characteristics in a population of elite soccer players, to search for correlations with the classical structural and electrocardiographic features of the athlete's heart and to describe the outcome at long-term follow-up.

Methods:

Study design and population

Overall, 338 male professional elite athletes, members of soccer clubs participating in the Italian national soccer championships, who underwent the first pre-participation screening between June 1980 and April 2008 at the Turin Institute of Sport Medicine were screened and retrospectively evaluated. Six subjects with negative T waves with a voltage >0.2 mV in more than 2 contiguous leads (one subsequently diagnosed with a mild form of hypertrophic cardiomyopathy) were excluded. The final cohort consisted of 332 subjects with an age between 15 and 37 years. The pre-participation screening consisted in the collection of a detailed clinical, physiological, familial, pharmacological and training history and in a complete physical examination. All subjects underwent a 12 lead ECG recording and a symptom-limited cardiopulmonary exercise test (Bruce protocol) with expired gas analysis. In subjects evaluated from 1989 to 1995, echocardiography was performed at discretion of the evaluating physician, afterwards all athletes underwent echocardiography.

ECG analysis

All electrocardiograms were retrospectively analyzed by two independent reviewers who were blinded to all subjects characteristics. In ambiguous cases final adjudication was achieved by consensus with a third reviewer.

All measurements were obtained with the use of 4x magnification lens. For all voltage measurements, the isoelectric line was considered to be the PR segment.

Heart rate, P wave duration, PR interval, QRS duration, QT interval were measured in lead II. In case of a J point elevation evident in lead II, QRS duration was measured in lead I. Correction of the QT interval was obtained using the Bazett's formula ($QTc=QT/\sqrt{RR}$). The presence of left atrial enlargement, first and second degree type I atrioventricular block, left anterior fascicular block, left posterior fascicular block, complete/incomplete, right and left bundle block was defined according to the AHA/ACCF/HRS recommendations for ECG interpretation[10-12]. QRS amplitude was recorded as a continuous variable using both the Sokolow-Lyon (S in V1 + R in V5 or V6 whichever greater) and the Cornell voltage index (R in aVL + S in V3). Voltage criteria for left ventricular hypertrophy were defined by the presence of at least one between the Sokolow-Lyon ≥ 35 mm and the Cornell voltage index ≥ 28 mm.

The ECG was deemed consistent with a J point elevation if an elevation ≥ 1 mm of the J point was evident in at least two contiguous leads. The height of J point elevation was measured in the lead showing the greatest elevation. The presence of J point elevation was categorized in each of the twelve leads separately and by territory (anterior [V1 to V3], inferior [II,III,aVF], lateral [V4 to V6, I and aVL]). In line with the original definition of ER[1,14,16], J point elevation in the anterior leads (from V1 to V3) was not excluded.

In subjects with J point elevation, the QRS-ST transition was classified into 3 groups according to its morphology in each lead. *ER without J wave* was defined by the combined presence of J point and ST segment elevation in the absence of any J wave. As previously reported[2,3], a *notched J wave* was defined by the presence of a sharp and well-defined hump immediately after the R-wave, while a *slurred J wave* was identified when the QRS-ST transition was characterized by a change in the slope of the terminal portion of the R wave (figure 1). To estimate the entity of J point elevation in this subgroup of subjects the J point elevation at the intersection between the tangents of the descending limb of the QRS complex and the J wave was measured.

The morphology of the ST segment was classified in *concave/rapidly ascending* (elevation of the ST segment >1 mm within 100 ms after the J point or after the change in slope of the terminal portion of the QRS complex) and

horizontal/descending (ST-segment elevation ≤1 mm within 100 ms after the J point or after the change in slope of the terminal portion of the QRS complex)[2].

Echocardiography

A complete echocardiographic assessment, performed during their first evaluation by a single experienced cardiologist, was available for 235 athletes. Echocardiography was performed in order to obtain cardiac chambers measurements and to exclude the presence of structural heart disease. Measurements of left atrial anteroposterior diameter, left ventricular (LV) wall thickness, LV systolic and diastolic diameters, volumes and ejection fraction were obtained in compliance with contemporary guidelines[13].

Follow up

For 269 (81.0%) athletes repeated clinical and electrocardiographic evaluations were available. The occurrence of events during follow-up was ascertained for all athletes looking through Turin's Institute of Sport Medicine archive, the soccer club's databases and the National soccer player Association online database. Follow-up started from the date of the first preparticipation screening and lasted until October 2012.

Statistical analysis

Descriptive analysis was performed using mean ± standard deviation for continuous variables and counts and percentages for categorical variables. Comparisons between groups were performed with the Student's t-Test, one way ANOVA and Yates corrected or uncorrected Chi-squared method when appropriate. All probability values were considered to be significant at a value ≤ 0.05. Only data of athletes who underwent echocardiographic examination were used for univariate and multivariable logistic regression analysis with a stepwise approach to identify characteristics associated with the presence of J point elevation. Using the entire population follow-up data, Kaplan Meier survival curves were plotted, stratifying by the presence/absence of J point elevation. All the analyses were performed with SPSS software (SPSS, Chicago, IL).

Results

Clinical characteristics of athletes are shown in table 1. Mean age at the time of ECG recording was 23.6±5.3 years; black athletes represented the 8.1% of the population. Average maximal oxygen consumption and maximal work load achieved during symptom-limited cardiopulmonary exercise test were respectively 53.5±11.0 ml/(kg min) and 237.7±41.4 Watts (W), consistent with a top level physical training for soccer players.

Two hundred and fourteen (64.4%) athletes showed an isoelectric J point (ISO J group) while in 118 (35.6%) a J point elevation ≥1 mm (ELE J group) at basal ECG was present. Excluding the anterior leads (from V1 to V3) any other type of J point elevation was evident in 85 (25.6%) subjects.

The presence of J point elevation was more frequent in black than in white athletes (62.9% vs. 33.1%; RR 1.901, 95% CI 1.366-2.647, p=0.003). No significant differences in terms of age, weight, height, body surface area were evident between athletes with and without J point elevation. Exercise capacity and maximal aerobic capacity did not significantly differ between athletes with and without J point elevation.

Electrocardiographic characteristics

Basal ECG parameters are shown in table 2. J point elevation was more frequently observed in subjects with slower heart rate (ISO J group: 59.8±13.6 bpm *vs.* ELE J group: 54.6±10.0 bpm, p <0,001) and increased voltage of the precordial leads evaluated with Sokolow Lyon index (ISO J group 30.3±9.1 *vs.* ELE J group 34.2±11.3, p<0,001; RR for positive Sokolow index 1.392 95% CI 1.045-1.853, p=0.034). Moreover, subject with J point elevation showed shorter QRS duration (ISO J group 85.1±9.7 ms *vs.* ELE J group: 82.5±9.4 ms, p=0.019) and shorter corrected QT interval (ISO J group 391.8±30.3 ms *vs.* ELE J group 384.4±29.1 ms, p=0.032).

In ELE J group, average J point elevation was 1.9±0.9 mm (range between 1 and 6 mm) and J point elevations greater than 3 mm were rarely found (5.1% of subjects, Fig.2, left panel).

Mean number of leads showing a J point elevation (including V1-V3) was 3.4±1.4 (range between 2 and 8, Fig. 2, right panel).

QRS-ST transition morphology

Distribution of different QRS-ST transition morphologies in ELE J group is shown in table 3.

Subjects with notched, slurred J waves and ER without J wave respectively showed a progressive lengthening of the QRS complex duration (notched J waves 78.8±8.8 ms; slurred J waves 81.3±8.3; ER without J wave: 85.7±9.9; p=0.005).

J point elevation distribution

Considering only athletes with a single morphology of the QRS-ST transition, 40 (33.8% of the ELE J group) subjects showed a J point elevation in the lateral (V4 to V6) leads, 26 (22.0% of the ELE J group) in the anterior (V1 to V3) leads, 17 (14.4% of the ELE J group) in the inferior (II, III and aVF) leads, 7 (5.9% of the ELE J group) both in the anterior and lateral leads and only 5 (1.5% of the ELE J group) in the inferior and lateral leads.

Considering QRS-ST transition in any single lead in the ELE J group (figure 3) ER without J wave was more common in the anterior leads, while the presence of a notched or slurred J waves was more frequently observed in lateral and inferior leads, respectively.

ST segment morphology

Ascending ST segment was present in 95 (80.5%) athletes while horizontal/descending in 23 (19.5%) in the ELE J group. In subjects with horizontal/descending ST segment 13 (3.9%) showed slurred J waves, 4 (1.2%) notched J waves while 6 (1.8%) both notched and slurred J waves. Considering the spatial distribution of J point elevation and J waves associated with a horizontal/descending ST segment, 13 athletes showed this pattern in the inferior leads (9 with a notched and 4 with a slurred J wave), 2 in the lateral leads (all notched) and 6 both in inferior and lateral leads (2 with notched J waves and 4 with slurred J wave in the inferior and notched J wave in the lateral leads). The combined presence of J point elevation, slurred J wave and horizontal/descending ST segment, recognized in the general population as a possible marker of arrhythmic risk, was rare, manifest only in 4 (1.2%) athletes.

Echocardiography

Complete echocardiographic data were available for 147 athletes of the ISO J group and for 88 of the ELE J group (Table 3). No significant differences were

observed between groups in terms of LV diameters, ejection fraction or left atrial anteroposterior diameter. Interventricular septum resulted significantly thicker in subjects with, compared to those without J point elevation (ISO J group 10.2±1.4 mm vs. ELE J group 10.8 ± 1.6 mm; p =0.002), while LV posterior wall thickness was almost significantly thicker in the former group compared to the latter (ISO J group 9.3±1.1 mm vs. ELE J group 9.6 ± 1.3 mm; p=0.06).

Univariate analysis revealed that black race, lower heart rate, Sokolow Lyon index and interventricular septum thickness were significantly associated with the presence of J point elevation. After adjusting for all these significant variables, lower heart rate (OR 0.968 95% CI 0.945- 0.993; p=0.011), increased interventricular septum thickness (OR 1.224 95%CI 1.014-1.478; p=0.036) and Sokolow Lyon index (OR 1.031 95% CI 1.002-1.060, p=0.033), but not black race (OR 0.424 95% CI 0.147-1.224; p=0.113), were significantly associated with J point elevation.

Follow up

Follow up data were available for the entire cohort of athletes. During follow-up (median 13,3 years; interquartile range 10,1-17,03 years) 2 deaths, due to non-cardiovascular reasons, were observed. One subject died at the age of 23 for acute leukemia. Another subject died in a road accident at the age of 28. In

none of the ECGs available for these two athletes a J point elevation was ever observed. Accordingly, no differences in the overall survival between subjects with and those without J point elevation were observed (p=0.294, fig.4).

Discussion

This retrospective analysis of a cohort of elite soccer players provided the opportunity to examine the clinical significance of J point elevation in the competitive sport setting, a field in which this ECG sign has been less investigated so far. The main findings of our study are: 1) the significant association between J point elevation and markers of left ventricular structural remodeling (e.g. increased interventricular septum thickness and Sokolow-Lyon index) as well as with lower resting heart rate; 2) after a long-term follow-up, no significant differences in survival were observed between athletes with and without J point elevation.

Previous studies recognized, in the general population or in non-professional athletes, the association between the presence of J point elevation and young age, male gender, black race, slower heart rate, increased voltage of the QRS complexes and enhanced aerobic fitness [9,14-17]. A recent work by Noseworthy et al. reported that, in a subgroup of football and rowing athletes, J point elevation on surface ECG was not significantly associated with any parameter

of structural remodeling typical of the athlete's heart, supporting the hypothesis that the evidence of J point elevation is a mere electrophysiological phenomenon and that it is not related to any form of exercise-induced left ventricular remodeling[9].

Our work has confirmed the association, already found in previous studies, between J point elevation, slower heart rate and increased QRS voltages in the precordial leads. Interestingly, a significant association between J point elevation and a thicker intraventricular septum was also observed both at univariate and multivariable analysis. The homogeneous sample of elite athletes under study, practicing the same type of high-intensity training, may have favored this observation. Posterior wall thickness was not considered as a covariate in multivariable analysis because not significant at univariate analysis. The presence of a mild form of left ventricular hypertrophy is a cornerstone of the "athlete's heart" and our findings suggest exercise induced left ventricular remodeling as the potential structural basis for J point elevation at surface ECG. Marked echocardiographic LV hypertrophy has been demonstrated in athletes of black ethnicity. However, only interventricular septum thickness but not black race was retained in our multivariable model, supporting the hypothesis of a mechanistic link between cardiac remodeling and J point elevation. A possible explanatory mechanism, based on animal studies[18] and sporadic case

reports in human, linking mild LV hypertrophy and QRS shortening was proposed by Boineau. According to this hypothesis an increased LV endocardial trabeculation and a greater endocardial invagination depth, may be associated with a deeper localization (into the mid myocardium) of the Purkinje fibers and may provide the anatomical basis for a faster activation of the thickened left ventricular walls[19]. The same mechanism was also recently proposed to explain the high prevalence of J point elevation in a population of patients with left ventricular non compaction cardiomyopathy[20]. An increased left ventricular trabeculation is associated with physical training. In fact, a recent MRI study comparing soccer players of European versus African descent reported a greater degree of myocardial trabeculation in the latter. According to in vitro studies[21], small gradients in ventricular repolarization (i.e. small differences in action potential duration in contiguous myocardial regions) exist in the normal left ventricle and are responsible for the J-point elevation; as this dispersion of repolarization increases in magnitude, increasingly taller J waves become manifest on the surface ECG. The presence of a mild form of ventricular trabeculation associated with hypertrophy may allow for a more rapid ventricular activation (evident as a shorter QRS complex) revealing the presence of J point elevation and J waves. QRS duration and coupling time between QRS complexes and J waves may influence both J point appearance and the morphology of the transition between QRS and ST segment. As a

matter of fact, in our population, the duration of QRS complex not only differed between subjects with and without J point elevation, but a trend for a progressively longer QRS complex was observed in subjects with notched, slurred J waves and early repolarization without J wave respectively.

Interestingly and apparently in contrast with previous studies conducted in non-professional athletes, in our population of elite athletes the presence of J point elevation was not associated with an increased aerobic fitness or maximal work load. This finding may be due to a ceiling effect in the incidence of J point elevation in highly trained athletes.

In our work the presence of a J point elevation was evident in more than 1 out of 3 professional top level soccer players: in this population, after a long-term follow-up no significant differences in survival were observed between athletes with and without J point elevation. This finding, should reassure Sport Medicine physicians performing pre-participation screening of athletes with unremarkable clinical history, avoiding unnecessary, time consuming and expensive subsequent evaluations.

Some limitations of our study should be acknowledged. The estimated risk of sudden cardiac death of subjects with J point elevation is very low, approximating 11 in 100000 over 10 years in the general population[8]. Even if the risk of sudden cardiac death is more than doubled in active compared with

sedentary subjects, the population included in the present study is too small to draw definite conclusions on the real prognostic impact of J point elevation in athletes. Nevertheless, to the best of our knowledge, to date this is the largest, long-term follow-up study investigating clinical features associated with J point elevation in elite athletes.

In conclusion, in our series of elite soccer players enrolled over 3 decades, more than one out of three showed a J point elevation confirming its high prevalence amongst physically active subjects. The correlation between J point elevation and increased interventricular septum thickness suggests a possible mechanistic role of exercise induced left ventricular hypertrophy in the genesis of early repolarization.

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Table 1. Clinical parameters

	Total Population N=332	Isoelectric J point N=214	Any J point elevation N=118	p-value
Mean age at first ECG, years	23.6±5.3	23.6±5.2	23.5±5.5	0.869
Anthropometrics				
Weight, kg	75.7±7.2	75.6±7.2	75.8±7.2	0.808
Height, cm	180.0±5.9	180.2±5.7	179.6±6.1	0.371
BSA, m^2	1.9±0.1	1.9±0.1	1.9±0.1	1
Race, n(%)				
White	305(91.9)	204(95.3)	101(85.6)	**0.003**
Black	27(8.1)	10(4.7)	17(14.4)	
Exercise Capacity				
VO2 max,(ml/(kg min))	53.5±11.0	53.3±12.3	54.0±8.4	0.582
Maximal work load, W	237.7±41.4	236.8±40.5	239.2±43.1	0.614

Table 2. Basal ECG parameters

ECG parameters	Total Population N=332	Isoelectric J point N=214	Any J point elevation N=118	p-value
Rhythm				
Sinus rhythm, n(%)	324(95,9)	208(97,2)	110(93,2)	
Atrial rhythm, n(%)	13(3.8)	5(2.3)	8(6.8)	0.150*
Junctional rhythm, n(%)	1(0.3)	1(0.5)	0(0)	
Heart rate, bpm	57.9±12.7	59.8±13.6	54.6±10.0	**<0.001**
P wave duration, ms	87.3±15.4	87.2±15.3	86.9±15.6	0.865
ECG criteria for left atrial enlargement, n(%)	1(0.3)	0(0)	1(0.8)	
PR interval, ms	163.8±27.2	161.5±24.9	166.3±29.0	0.114
AV conduction*				
Normal, n(%)	320(94.7)	206(96.3)	110(93.2)	0.236†
I degree AV block, n(%)	16(4.7)	7(3.3)	7(6.0)	
II degree type 1 AV block, n(%)	1(0,3)	0(0)	1(0.8)	
QRS duration, ms	84.3±9.7	85.1±9.7	82.5±9.4	**0.019**
QRS axis, °	65.1±24.1	64.8 ±23.9	66.2±23.5	0.608
Not determinable	4(1.2)	0(0)	1(0.8)	
Intraventricular conduction defect, n (%)	15(4.5)	9(4.2)	5(4.2)	0.786‡
Anterior fascicular block, n(%)	2(0.6)	0(0)	1(0.9)	
Posterior fascicular block, n(%)	1(0.3)	0(0)	1(0.9)	
Complete RBBB, n(%)	1(0.35)	1(0.5)	0(0)	
Incomplete RBBB, n(%)	11(3.3)	8(3.7)	3(2.5)	
Complete LBBB, n(%)	0(0)	0(0)	0(0)	
QT interval, ms	401.0±34.8	397.3±35.9	406.1±31.5	**0.026**
Corrected QT interval, ms	389.3±30.0	391.8±30.3	384.4±29.1	**0.032**
Sokolow Lyon index, mm	31.8±10.2	30.3±9.1	34.2±11.3	**<0.001**
Positive Cornell Voltage index, n(%)	22(6.5)	16(7.5)	4(3.4)	0.209
Negative T waves, n(%)	6 (1.8)	/	/	

* sinus rhythm (SR) vs other than SR

† Normal AV conduction *vs*. first and second degree type 1 AV block

‡ Normal *vs*. intraventricular conduction defect

Table 3. Echocardiographic parameters.

Characteristics	Isoelectric J point N=147	Any J point elevationN=88	p-value
Interventricular septum thickness, mm	10.2±1.4	10.8±1.6	**0.002**
Posterior wall thickness, mm	9.3±1.1	9.6±1.3	0.06
LVEDD, mm	54.8±4.1	54.4±4.0	0.466
LVESD, mm	35.3±3.4	34.8±3.6	0.287
EF, %	64.2±3.2	64.3±4.5	0.843
Left atrial AP diameter, mm	35.2±3.9	35.4±4.4	0.717
Left ventricular mass/BSA, g/m^2	106.8±19.8	111.1±20.6	0.113

Table 4. Morphologies of the QRS-ST transition.

		Isoelectric J point N(%)
Single		95
	ER without J wave	53(44.9%)
	Notched J waves	34(28.8%)
	Slurred J waves	8(6.8%)
Combined		23
	Notched + Slurred J waves	7(5.9%)
	Notched + ER without J wave	12(10.1%)
	Slurred + ER without J wave	1(0.8%)
	Notched+ Slurred + ER without J wave	3(2.5%)

Figure 1: Morphologies of the QRS-ST transitions: A) early repolarization without J wave; B) notched J wave with ascending ST segment; C) notched J wave with horizontal/descending ST segment; D) slurred J wave with ascending ST segment; E) slurred J wave with horizontal/descending ST segment.

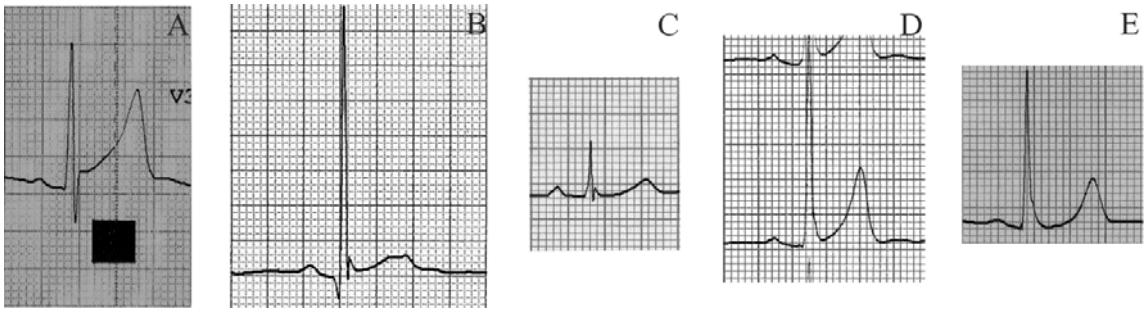

Figure 2. Left panel: distribution of the amplitudes of J point elevation. Right panel: distribution of the number of leads with a J point elevation ≥1 mm.

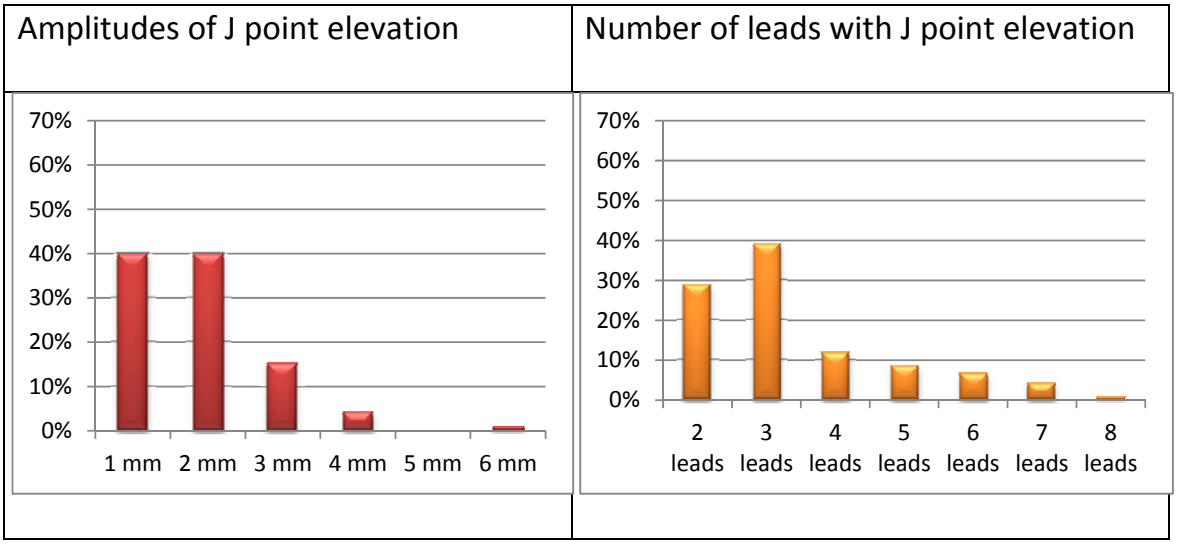

Figure 3. Distribution of the different QRS-ST transition morphologies in different leads.

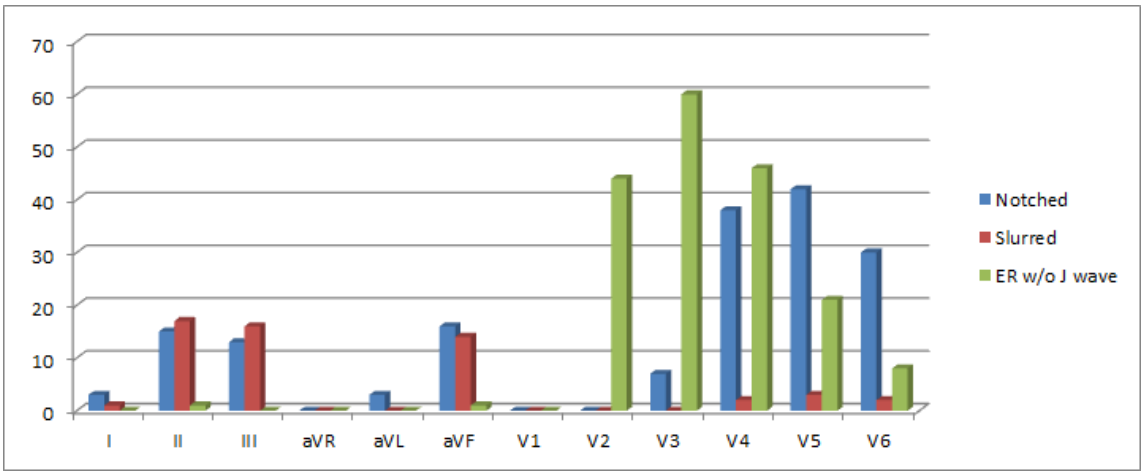

Figure 4. Kaplan Meier survival curves of athletes with and without J point elevation.